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EFFECTS OF SEPTAL AND HYPOTHALAMIC
LESIONS ON SHIVERING

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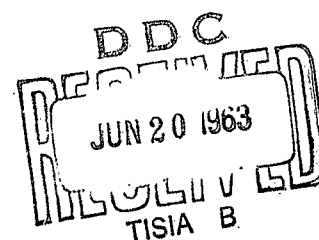
September 1962

ARCTIC AEROMEDICAL LABORATORY

AEROSPACE MEDICAL DIVISION
AIR FORCE SYSTEMS COMMAND
FORT WAINWRIGHT, ALASKA

Project 8238-22

(Prepared under Contract AF41(657)-344 by
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ABSTRACT

Shivering and heat loss in the cold were determined in 46 cats several weeks or months after bilateral destruction of various septal and hypothalamic regions. Septal lesions had no effect on either parameter. The tremor was abolished or markedly reduced in cats with lesions in the dorsomedial region of the posterior hypothalamus, but postural, pilomotor and behavioral responses to cooling persisted. Lesions of the dorsolateral region of the posterior hypothalamus increased heat loss despite the presence of shivering, huddling and piloerection. These results confirmed our previous electrical stimulation data that the primary region controlling the efferent (motor) aspect of shivering is the dorsomedial region of the posterior hypothalamus and additionally indirectly suggested that the dorsolateral region of the posterior hypothalamus is implicated in cold-induced cutaneous vasoconstriction.

PUBLICATION REVIEW

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EFFECTS OF SEPTAL AND HYPOTHALAMIC LESIONS ON SHIVERING *

SECTION 1. INTRODUCTION

On the basis of electrical stimulation experiments (Stuart et al, 1962; 1961a) we recently reported that the dorsomedial region of the posterior hypothalamus and the posterior septal region of the forebrain were implicated in the production of shivering. A greater intensity of stimulus was needed to produce more latent and less intense shivering during septal than during hypothalamic stimulation. This suggested that septal influences were secondary to a primary hypothalamic control of the tremor. However, there are reports of shivering being abolished in acute (Jacobson et al, 1960) and chronic (Jacobson and Squires, 1961) cats with lesions that include the posterior septum and of profound hypothermia (Bond et al, 1953) following such lesions. Additionally, while other lesion studies have suggested the posterior hypothalamus is essential for shivering, there is no agreement among these investigations as to the specific region of the posterior hypothalamus so involved (Bazett et al, 1933; Birzis and Hemingway, 1956; Blair and Keller, 1946; Clark et al, 1939; Frazier et al, 1936; Keller, 1935; Keller and Hare, 1932). In view of such controversy, we studied the effects of septal and hypothalamic lesions on shivering in unanesthetized cats several weeks or months after surgery.

SECTION 2. METHODS

In 32 cats anesthetized with pentobarbital (35 mg/kg, i.p.) bilateral electrolytic lesions were made in various hypothalamic regions, and in 14 cats lesions were made in the septal region of the forebrain. Postoperative care of each animal was given special attention. The rectal temperature was kept at 36° to 38° C by appropriate alterations in environmental temperature. For the first one to two weeks after surgery each animal was tube fed, with frequent testing for the recovery of licking and swallowing reflexes. Care was taken to keep the animals warm and dry, and they were given daily periods of exercise. When they had regained their preoperative weight by spontaneous eating, their responses to cold stresses were studied.

* Submitted for publication October 1961 and held by Contract Monitor.

The intensity of shivering, if any, was gauged by the ratio of oxygen consumption rate (OCR) while exposed to an environment of 0° to 5° C air and while resting and not shivering in an environment of 25° to 30° C air. Heat loss was estimated by the ratio $\Delta T_r/\Delta t$ in °C/min, where ΔT_r is the drop in rectal temperature while exposed to 0° to 5° C air from time Δt . The apparatus and procedure for measuring these parameters have been described previously (Stuart et al, 1961b). As a control, similar determinations were made in nine intact cats. If the animals with lesions had OCR shivering/resting and $\Delta T_r/\Delta t$ values that fell within the control range, they were sacrificed immediately after testing, their brains fixed in formalin and sectioned every 80 μ , alternate sections being stained with buffered thionine. If they did not have control responses they were tested at least twice more at varying lengths of time after the first test before being sacrificed.

SECTION 3. RESULTS

Controls

Table I lists the mean ratios of OCR shivering/resting and $\Delta T_r/\Delta t$ for the nine intact cats. Both these parameters can vary in intact cats over rather wide ranges and relatively independently of each other (Stuart, 1961). Therefore, it was decided to compare the results for the animals with lesions to the mean and range of data presented in Table I rather than to data collected on each animal prior to surgery. It was felt that a decline in shivering intensity would only be significant if the ratio of OCR shivering/resting was below 2.0 and in $\Delta T_r/\Delta t$ if greater than 0.07° C/min, i. e. values beyond the ranges listed for intact cats in Table I.

Septal Lesions

Eight of the animals with septal lesions were given cooling tests. Of those not tested one died 6 days, one 10 days, and one 34 days after surgery. In two other animals electrodes were inserted into the septum but no current was passed. These two animals recovered within a week but were not subjected to cooling. Table II, part A lists the values for OCR shivering/resting and $\Delta T_r/\Delta t$ of three of the cats that were tested. The extent of their lesions are shown schematically in Figure 1. The number of cats with similar lesions and similar results is also indicated in Table II, part A. Both the intensity and shivering and heat loss in the cold were normal in all these animals. No differences were observed in animals with medial, lateral or complete

TABLE I

Shivering intensity and heat loss of nine intact cats*

	<u>OCR S/R</u>	<u>$\Delta T_r/\Delta t$</u>
Mean	2.7	0.04
Range	2.1-3.8	0.02-0.07
S.D.	0.5	0.02

*OCR S/R, ratio of oxygen consumption rate while shivering in 0° to 5° C air and while resting and not shivering in 25° to 30° C environment; $\Delta T_r/\Delta t$, drop in rectal temperature (ΔT_r) while exposed to 0° to 5° C air for time Δt in $^{\circ}\text{C}/\text{min}$; S.D., standard deviation of the mean.

TABLE II

Shivering intensity and heat loss of cats with representative septal and hypothalamic lesions*

<u>Cat No.</u>	<u>Lesion</u>	<u>Days after Surgery</u>	<u>OCR S/R</u>	<u>$\Delta T_r/\Delta t$</u>
A. Septal Lesions				
S13 - (2)	PS-T	40	2.7	0.03
S6 - (4)	PS-L	50	2.3	0.02
S11 - (2)	PS-M	26	3.7	0.03
B. Hypothalamic Lesions				
H1 - (3)	AH-L	48	2.1	-0.01
H9 - (4)	PH-L	46	2.5	0.20
F8 - (2)	PH-DL	46	2.6	0.13
ST38 - (6)	PH-VL	31	3.2	0.03
H13	PH-DM	21	1.0	0.11
H18 - (2)	PH-DM	91	1.2	0.10

* (), number of cats with similar lesions and results; PS, posterior septum; AH, anterior hypothalamus; PH, posterior hypothalamus; T, total; L, lateral; M, medial; DL, dorsolateral; VL, ventrolateral; DM, dorsomedial; Days after Surgery, number of days between surgery and test; other abbreviations as in Table I.

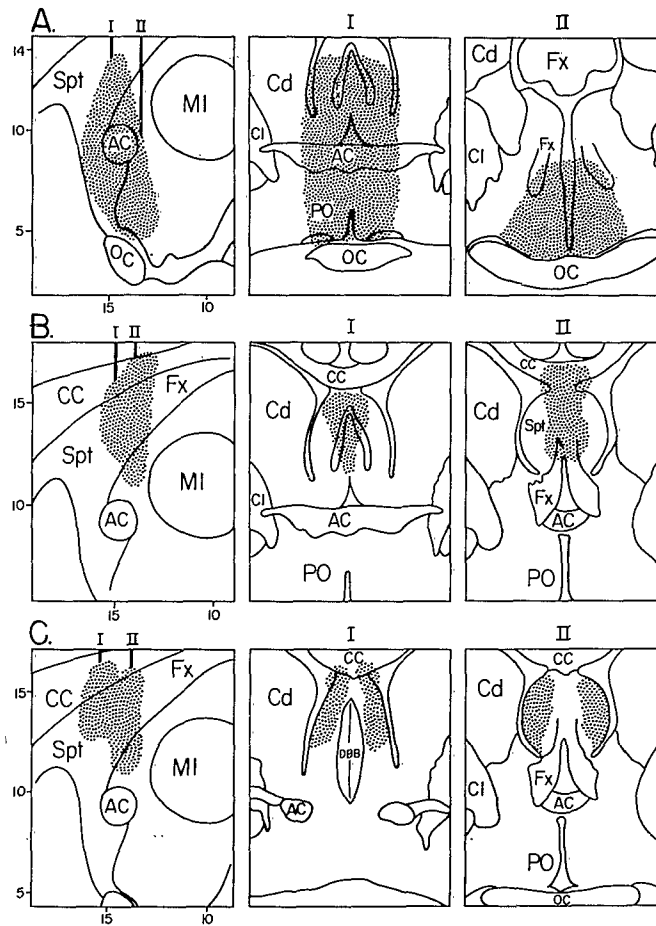


FIGURE 1

Midsagittal and frontal plane representations of typical septal lesions. Extent of minimum bilateral tissue destruction indicated in shaded area. Numbers on mid-sagittal schemata indicate mm anterior and dorsal to interauricular line. A, total destruction of the posterior septum and preoptic region in cat S13; B, medial posterior septal lesion in cat S11; C, lateral posterior septal lesion in cat S6. Abbreviations: AC, anterior commissure; CC, corpus callosum; CD, caudate nucleus; CI, internal capsule; Fx, fornix; OC, optic chiasm; M, mammillary body; MI, mass intermedia; PO, preoptic area; Spt., septum.

posterior septal lesions. In one of the latter group (S13, Figure 1-A), both posterior septal and preoptic regions were destroyed to sever anterior septal projections to the hypothalamus.

Hypothalamic Lesions

Twenty-four of the animals with hypothalamic lesions recovered from surgery. One animal died 2 hours, two 5 days, one 8 days, and one 12 days after surgery. Three animals died in the course of heat stress tests administered five days after surgery. Of the 24 animals that were tested, six typify the results and Table II, part B, lists the measurements that were made on these animals. Nineteen of these cats shivered with normal intensity. Their various lesions included all anterior and posterior hypothalamic tissue except the dorsomedial region of the posterior hypothalamus. The five cats that did not shiver at normal intensity had lesions abolishing or encroaching upon this region. In two of these cats, dorsomedial destruction was incomplete and shivering was only mildly reduced, the ratio of OCR shivering/resting being 1.9 and 1.8 when tested 33 and 46 days after surgery, respectively. Two others had more complete destruction of this region (Figure 2, A and B) and shivering was markedly reduced, the OCR shivering/resting being 1.2 and 1.4, respectively, when tested 91 days after surgery. In these two animals, shivering was feeble, intermittent and restricted to the neck and proximal musculature of the forelimbs. In one cat, with dorsomedial posterior hypothalamic destruction (Figure 2, C), shivering was completely abolished till sacrificed, 40 days after surgery. Twenty-one days after surgery the ratio of OCR shivering/resting was 1.0. Forty days after surgery when moving pictures were taken of this animal's ability to piloerect and huddle when not shivering at a rectal temperature of 33° C, it was additionally observed that it could make appropriate behavioral responses to cooling; i. e., when cold and not shivering the cat actively sought a warm pad and heating lamp as they were moved about the laboratory.

In six cats shivering was normal but $\Delta T_r/\Delta t$ while shivering in 0° to 5° C air was excessive. Despite this increase in heat loss, pilomotor and postural responses to cooling were evident. In three of these cats the lesions involved the lateral posterior hypothalamus (Figure 3, A), in two others the dorsolateral posterior hypothalamus (Figure 3, B and C), and in one all the posterior hypothalamus except the dorsomedial region on one side. This latter animal did not appear to huddle and piloerect in the cold as effectively as the others. Three cats with large lateral lesions in the anterior hypothalamus, and six others with ventrolateral lesions in the posterior hypothalamus, had normal $\Delta T_r/\Delta t$ values.

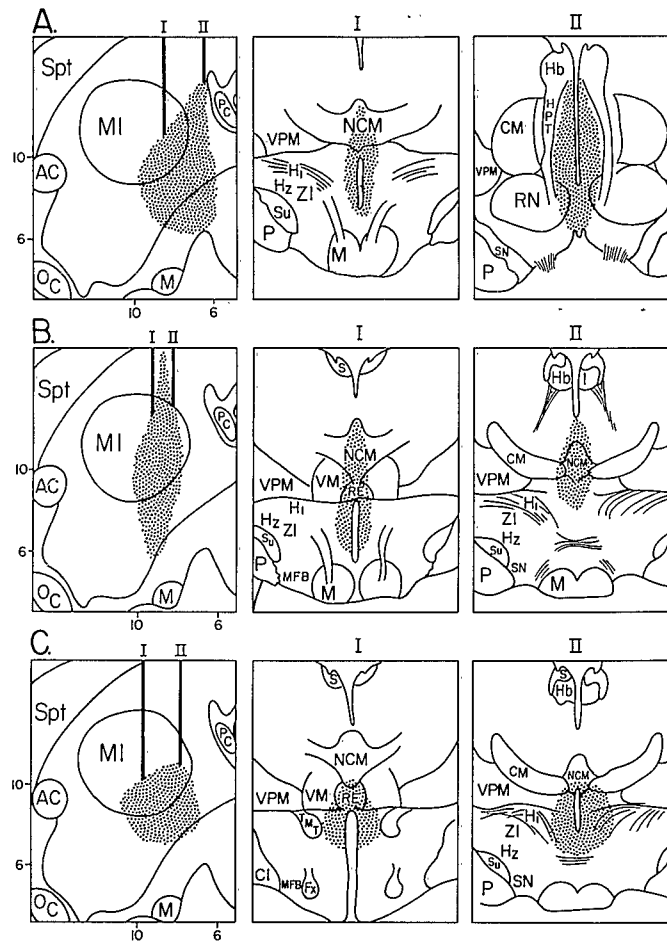


FIGURE 2

Dorsomedial posterior hypothalamic lesions markedly reducing (A, cat H18; B, cat H19) or abolishing (C, cat H13) shivering. Abbreviations: CM, centromedian nucleus of thalamus; $H_{1,2}$ Forel's fields; Hb, habenular nucleus; HPT, habenulo-peduncular tract; MFB, medial forebrain bundle; NCM, centromedian nucleus of thalamus; P, cerebral peduncle; PC, posterior commissure; RE, reuniens nucleus; RN, red nucleus; S, stria terminalis; SN, substantia nigra; Su, subthalamic nucleus; TMT, mammillo-thalamic tract; VM, ventromedial nucleus of thalamus; VPM, ventro-posteromedial nucleus of thalamus; ZI, zona incerta. Other abbreviations as in Figure 1.

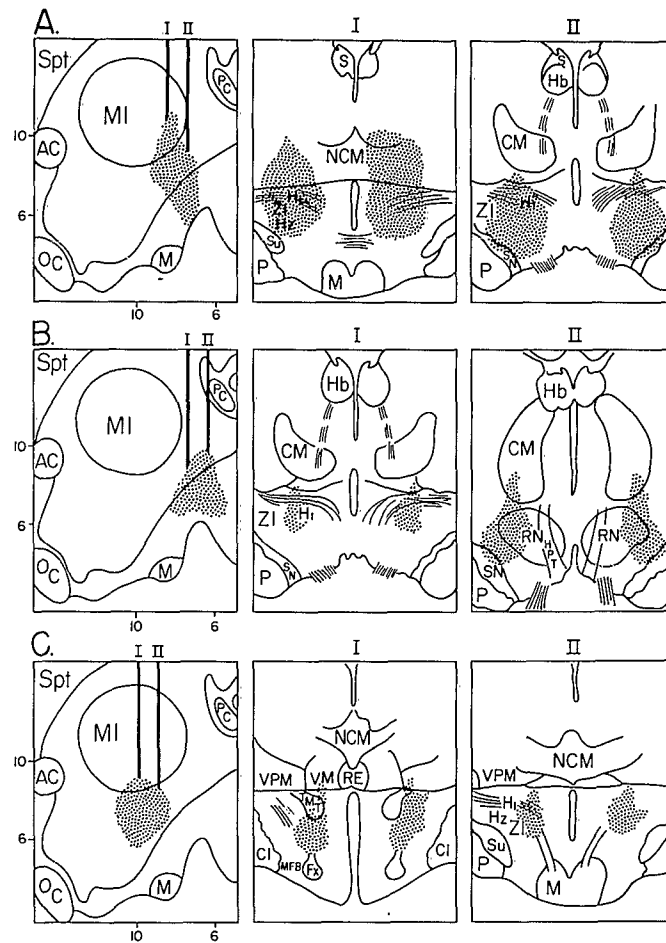


FIGURE 3

Dorsolateral posterior hypothalamic lesions reducing heat retentive capacity without affecting the intensity of shivering. A, cat F10; B, cat F8; C, cat H15. Abbreviations as in Figures 1 and 2.

In the early postoperative period the animals were sometimes removed from their controlled environmental temperature and rectal temperature measured after several hours in a 20° to 25° C environment. Such tests showed that animals with dorsomedial lesions in the posterior hypothalamus recovered temperature regulation in this room temperature environment 7 to 14 days earlier than animals with lateral or dorsolateral lesions of the posterior hypothalamus.

Locomotor Disturbances. In cats with hypothalamic lesions spontaneous movements were reduced for the first two to three weeks after surgery. They made no attempts to escape when their cage doors were left open but rather assumed huddled and often bizarre postures for many hours. These odd postures could hardly be termed "catatonic" in that the animals resisted the investigators' attempts to place them in equally bizarre positions. They resumed licking reflexes the day after surgery and vigorously chewed and swallowed food placed in their mouths. They did not eat spontaneously until the second to third week after surgery, at which time they responded alertly to visual, auditory and nociceptive stimuli. The cats with septal lesions were also hypokinetic in the early postoperative period but when compelled to move, had normal locomotor control. They did not assume bizarre postures. They were capable of chewing and swallowing food placed in their mouths but would not eat spontaneously in this early postoperative period.

SECTION 4. DISCUSSION

Septal Influence on Shivering

In a recent review of telencephalohypothalamic relations, Gloor (1956) concluded that the limbic system of the telencephalon "is not critically involved in the integration of the very same somato-autonomic mechanisms which it is apt to influence on electrical stimulation..." He found no reports of an impairment in the integration of basic autonomic and somatomotor mechanisms following limbic lesions. Our results concur in that electrical stimulation of a limbic structure, the posterior septum, was shown to affect shivering (Stuart et al, 1962; 1961a) but there was no measurable alteration in the metabolic effectiveness of shivering following destruction of part or all of this forebrain region. Such results conflict in several respects with those of Bond and coworkers (1953), who reported coma, hyperglycemia, hypothermia and often spontaneous death in cats with lesions involving the posterior septum. Despite aseptic surgery the majority of their animals died five days after surgery. Of the 16 cats that lived 12 days or longer after surgery, only two were hypothermic. Thus it is difficult to compare their

results to the present ones, since our cats with similar lesions were not exposed to cooling until at least 26 days after surgery. Similarly Jacobson, Craig and Squires (1960; Jacobson and Squires, 1961) have recently reported an impairment in shivering in the early postoperative period after preoptic lesions that included the ventral margin of the posterior septum. As discussed previously (Stuart et al, 1961a), conclusions based on functional deficits immediately following electrolytic destruction of any region of the brain must be interpreted with caution. Such deficits are always greater in the early postoperative period than would be expected at a later time.

The present results do not refute the possibility that regions rostral to the hypothalamus, such as the septum, play a role in the modulation of shivering in the intact animal. The production and suppression of shivering by hypnotic suggestion (Gessler and Hansen, 1927) and the elevation or depression of heat production induced by classical Pavlovian conditioning (Bykov, 1957) might well involve telencephalic projections to the hypothalamus (Stuart, 1961).

Hypothalamic Activation of Shivering

The results confirm our previous electrical stimulation data (Stuart et al, 1962; 1961a) in implicating the dorsomedial region of the posterior hypothalamus in the production of shivering. In one animal shivering was completely abolished by destruction of this region up to 40 days after surgery, at which time it was sacrificed. In two other cats with similar but more restricted lesions, shivering was remarkably reduced in intensity and restricted to neck and proximal limb musculature up to thirteen weeks after surgery. It might be argued that this feeble shivering was indicative of the "plasticity" of the brain or its ability to subserve shivering by a reorganization of pathways after destruction of the normal pathway. If correct, posterior hypothalamic tissue would still be involved, since Bard (1961) has maintained decerebrate cats for many months after surgery without observing a return of shivering. In one cat of the present study, all posterior hypothalamic tissue except the ipsilateral dorsomedial region was destroyed without affecting the intensity of shivering. The lesions in the animals in which there was a return of feeble shivering were not as complete in their rostro-caudal extent as the lesion in the cat in which shivering was abolished. Additionally, in two other cats with partial bilateral destruction of this region, shivering was subnormal but not as reduced as that in animals with more complete destruction of the dorsomedial region. Thus, it seems more likely that the feeble and intermittent shivering observed in the cats in question was due to incomplete destruction of the dorsomedial region of the posterior hypothalamus.

Shivering, long known to be suppressed during localized warming of the pre-optic anterior hypothalamic region (Hemingway et al, 1940; Magoun et al, 1938), has recently been evoked by cooling the same region with little or no fall in skin temperature (Andersson and Larsson, 1961; Chatonnet, 1961; Hammel et al, 1960). This suggests but, in the absence of sufficient microelectrode experimentation, not altogether proves that brain temperature is "sensed" in this discrete prosencephalic region and that shivering can be evoked by its thermal activation without the support of a peripheral input from cutaneous "cold" fibers. Similarly, it seems equally as evident that shivering can be evoked by lowering skin temperature with little or no change in brain temperature (Benzinger et al, 1961; Chatonnet, 1961). In one cat from the present results (S13) vigorous shivering was present after destruction of pre-optic anterior hypothalamic tissue including the allegedly thermally sensitive area. This in no way conflicts with the reports of shivering induced by cooling this region if it is accepted that a cold skin can evoke the tremor. However, the absence of shivering in cats with dorso-medial posterior hypothalamic lesions does not imply that this latter region is a "center" for the hypothalamic reception of information from the cutaneous "cold fibers" because animals with destruction of this region could still huddle, piloerect and make appropriate behavioral responses while not shivering in the cold. Thus, it appears that the reception of peripheral thermal information is quite diffuse within the hypothalamus and that the dorsomedial region of the posterior hypothalamus is implicated in the efferent (motor) aspect of shivering. Further, it is not implied that this region of the hypothalamus is implicated solely in shivering, but rather that activation of certain neurons within this region produces shivering in addition to other ergotropic effects (Hess, 1957; Ranson and Magoun, 1939; Stuart, 1961).

Separation of Hypothalamic Regions Involved In Heat Production and Heat Retention

Destruction of the dorsolateral region of the posterior hypothalamus resulted in increased heat loss but not heat production. However, in such animals postural and pilomotor responses to cooling persisted, thereby suggesting that cutaneous vasoconstriction was reduced in the cold. This may explain some of the diverse conclusions that have appeared in the literature concerning the neurogenesis of temperature regulating mechanisms against cooling. It is attractive to speculate that in the early postoperative period the animal with lateral or dorsolateral posterior hypothalamic lesions is hypothermic at room temperature by virtue of impaired cutaneous vasoconstriction, the diminished heat production being due, not to the loss of neural tissue, but to generalized debilitation following surgery. Conversely, the animal with dorsomedial posterior hypothalamic destruction quickly

recovers from hypothermia at room temperature due to the integrity of the lateral hypothalamic tissue subserving heat retentive mechanisms which are "physical," and hence less affected by postoperative trauma.

Other Deficits

Deficits in feeding behavior (being able to eat and not attempting to) and escape behavior were present in the early postoperative period following both septal and posterior hypothalamic lesions but are not unique to these regions. Such deficits have also been observed following subthalamic lesions (Adey and Lindsley, 1959), lemniscal lesions of the lower midbrain (Stuart, 1961), and pallidal lesions (Buchwald, personal communication). Perhaps these disturbances were related more to postoperative depression than to the specific tissue destroyed. The assumption of bizarre postures following posterior hypothalamic lesions was most striking and in keeping with the integrative effects exerted by the hypothalamus on motor as well as visceral activity (Jacobson et al, 1960). Even this pronounced deficit regressed within three postoperative weeks in sharp contrast to deficits in shivering and heat retentive capacity following dorsomedial and dorsolateral posterior hypothalamic lesions that could still be quantitatively measured long after all other apparent disturbances had abated.

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<p>Arctic Aeromedical Laboratory, United States Air Force (AFSC), APO 731, Seattle, Wash. Rpt. AAL-TDR-62-18. EFFECTS OF SEPTAL AND HYPOTHALAMIC LESIONS ON SHIVERING. Sept. 1962. 14 p incl. tables, illus., 28 ref. Unclassified Report</p> <p>Shivering and heat loss in the cold were determined in 46 cats several weeks or months after bilateral destruction of various septal and hypothalamic regions. Septal lesions had no effect on either parameter. The tremor was abolished or markedly reduced in cats with lesions in the dorsomedial region of the posterior hypothalamus, but postural, pilomotor and behavioral responses to cooling persisted. Lesions of the dorsolateral region of the posterior hypothalamus increased heat loss despite the presence of shivering, huddling and piloerection. These results confirmed our previous electrical stimulation data that the primary region controlling the efferent</p>	<ol style="list-style-type: none"> 1. Shivering 2. Hypothalamus 3. Cats I. Project 8238-22 II. Contract AF41(657)-344 III. Univ. of California at Los Angeles IV. Stuart, D. G. Y. Kawamura, A. Hemingway, W. M. Price V. Available from OTS VI. In ASTIA collection 	<p>(motor) aspect of shivering is the dorsomedial region of the posterior hypothalamus and additionally indirectly suggested that the dorsolateral region of the posterior hypothalamus is implicated in cold-induced cutaneous vasoconstriction.</p>
<p>Arctic Aeromedical Laboratory, United States Air Force (AFSC), APO 731, Seattle, Wash. Rpt. AAL-TDR-62-18. EFFECTS OF SEPTAL AND HYPOTHALAMIC LESIONS ON SHIVERING. Sept. 1962. 14 p incl. tables, illus., 28 ref. Unclassified Report</p> <p>Shivering and heat loss in the cold were determined in 46 cats several weeks or months after bilateral destruction of various septal and hypothalamic regions. Septal lesions had no effect on either parameter. The tremor was abolished or markedly reduced in cats with lesions in the dorsomedial region of the posterior hypothalamus, but postural, pilomotor and behavioral responses to cooling persisted. Lesions of the dorsolateral region of the posterior hypothalamus increased heat loss despite the presence of shivering, huddling and piloerection. These results confirmed our previous electrical stimulation data that the primary region controlling the efferent</p>	<ol style="list-style-type: none"> 1. Shivering 2. Hypothalamus 3. Cats I. Project 8238-22 II. Contract AF41(657)-344 III. Univ. of California at Los Angeles IV. Stuart, D. G. Y. Kawamura, A. Hemingway, W. M. Price V. Available from OTS VI. In ASTIA collection 	<p>(motor) aspect of shivering is the dorsomedial region of the posterior hypothalamus and additionally indirectly suggested that the dorsolateral region of the posterior hypothalamus is implicated in cold-induced cutaneous vasoconstriction.</p>